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Cognitive Prediction of Reading, Math, and Attention: Shared and Unique Influences

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Abstract

The current study tested a multiple-cognitive predictor model of word reading, math ability, and attention in a community-based sample of twins aged 8 to 16 years ($N = 636$). The objective was to identify cognitive predictors unique to each skill domain, as well as cognitive predictors shared among skills that could help explain their overlap and thus help illuminate the basis for comorbidity of related disorders (reading disability, math disability, and attention deficit hyperactivity disorder). Results indicated that processing speed contributes to the overlap between reading and attention as well as math and attention, while verbal comprehension contributes to the overlap between reading and math. There was no evidence that executive functioning skills help account for covariation among these skill domains. Instead, specific executive functions differentially related to certain outcomes (i.e., working memory to math and inhibition to attention). We explored whether the model varied in younger versus older children and found only minor differences. Results are interpreted within the context of the multiple deficit framework for neurodevelopmental disorders.

Introduction

Understanding the basis of the comorbidity among neurodevelopmental disorders has been a very active area of research in recent years. One of the most rigorously examined phenotypic associations is the comorbidity between reading disability (RD, or dyslexia) and Attention Deficit Hyperactivity Disorder (ADHD), for which there is now evidence of both shared

cognitive and genetic risk factors (McGrath et al., 2011; Willcutt et al., 2010). However, the association between RD and ADHD is not exclusive; various other comorbidities have been identified in children, including reading disability and math disorder (MD, or dyscalculia) (Landerl & Moll, 2010), as well as math disorder and ADHD (Capano, Minden, Chen, Schacher, & Ickowicz, 2008). Rarely, though, have all three symptom dimensions been studied in a large enough sample of children to allow for an investigation of the underlying cognitive relationships among them. The main goal of this study is to advance these important lines of inquiry by modeling the shared and unique cognitive risk factors that predict reading, math, and attention using data from a large community-based sample of twins from the Colorado Learning Disabilities Research Center (CLDRC).

How these three skill dimensions, and their associated disordered states, relate to one another at cognitive and etiologic levels of analysis is a central issue in the field of developmental neuropsychology, with clinical and theoretical implications. Apparent comorbidity among neurodevelopmental disorders can arise for various artifactual reasons, such as referral biases, definitional overlap, or assortative mating. Another possible explanation is a phenocopy phenomenon, in which symptoms of one disorder lead to secondary expression of the symptoms in another disorder. For example, perhaps children with poor attention struggle with academic skills not because they have a primary learning disability but because they are not able to benefit from instruction to the same extent as their peers. Previous research suggests that these artifactual explanations are unlikely to fully account for overlap among the symptom dimensions that are the focus of this paper (Pennington, Willcutt, & Rhee, 2005).

The theoretical model that guides the current study is the multiple cognitive deficit model of neurodevelopmental disorders (Pennington, 2006). In this investigation, we extend this deficit-focused model to account for individual differences across the full range of abilities. According to the multiple deficit framework, each symptom dimension can be predicted by several underlying cognitive factors. Genuine comorbidity among symptom dimensions or disorders arises because some of the underlying cognitive skills are shared by disorders. The overlap at the symptom level is less than 100% because each dimension also has unique predictors. This model has been applied successfully to several sets of symptom dimensions or disorders (Archibald, Cardy, Joanisse, & Ansari, 2013; Carroll & Myers, 2010; Christopher et al., 2012). In the sections that follow, we will provide a brief overview of the various two-way comorbidities embedded in this study. Subsequently, we will outline a rationale for the proposed set of hypotheses that we will test in this dataset in order to understand more clearly the relations among reading, math, and attention.

RD and ADHD

Although RD and ADHD each occur in approximately 5–10% of children in the population, 15 to 30% of children with either RD or ADHD also meet criteria for the other disorder, a higher-than-chance comorbidity (Willcutt et al., 2012). Research suggests that these disorders, along with virtually all other behaviorally defined disorders, represent the low tail of ability in a normal distribution (Pennington, 2014; Rodgers, 1983). Thus, the categorical diagnoses represent somewhat arbitrary cut-offs in continuous variables.

Twin studies have shown a significant genetic association between RD and ADHD, particularly between single word reading and the inattentive symptom dimension (Willcutt, Pennington, Olson, & DeFries, 2007). Neuropsychological studies have sought to identify cognitive deficits unique to each disorder. In the RD literature, there is ample support for a deficit in phonological awareness (PA) and other aspects of phonological processing (Boada & Pennington, 2006; Olson, Forsberg, & Wise, 1994; Vellutino, Fletcher, Snowling, & Scanlon, 2004). However, weaknesses have been identified in several other cognitive domains, such as broader speech and language processing (Bishop & Adams, 1990), naming speed (NS) (Compton, Olson, DeFries, & Pennington, 2002; Purvis & Tannock, 2000; Semrud-Clikeman, Guy, Griffin, & Hynd, 2000; Wolf & Bowers, 1999), processing speed (PS) (Caravolas, Volin, & Hulme, 2005; Catts, Gillispie, Leonard, Kail, & Miller, 2002; Kail & Hall, 1994; Shanahan et al., 2006; Willcutt, Pennington, Olson, Chhabildas, & Hulslander, 2005), and verbal working memory (VWM) (Rucklidge & Tannock, 2002; Swanson, Mink, & Bocian, 1999; Willcutt et al., 2001; Willcutt et al., 2005).

In the case of ADHD, there is less agreement about core neuropsychological deficits. Various aspects of executive functioning have been implicated, including response inhibition (Barkley, 1997), and organization/planning and working memory (WM) (Willcutt et al., 2005). Deficits in PS (Shanahan et al., 2006; Willcutt et al., 2005), NS (Rucklidge & Tannock, 2002; Shanahan et al., 2006; Willcutt et al., 2005), and reaction time variability (Kuntsi & Klein, 2012) have also been reported.

Our group has previously investigated the cognitive and etiologic overlap of RD and ADHD. Shanahan et al. (2006) showed that PS was a likely shared cognitive deficit that could account for the comorbidity between these disorders. Extending this work, McGrath et al. (2011) tested a multiple cognitive deficit model of RD and ADHD using multiple latent cognitive factors to predict three latent symptom dimensions (word reading, attention, and hyperactivity/impulsivity). Each symptom dimension had several significant predictors. PA, NS and PS predicted reading, while inhibition and PS predicted inattention and hyperactivity/impulsivity. PS was the only cognitive predictor shared by all three symptom dimensions. With PS in the model, the covariance between reading-attention and reading-hyperactivity/impulsivity became nonsignificant. Thus, the authors concluded that PS primarily accounted for the significant correlation (i.e., comorbidity) between the two symptom dimensions.

PS has also been shown to share common genetic influence with RD and ADHD symptom dimensions. Willcutt et al. (2010) used a Cholesky decomposition analysis in the CLDRC twin sample to estimate the shared and independent genetic influences on reading, inattention, hyperactivity/impulsivity, and PS and NS. After accounting for genetic influences shared by PS, reading, inattention, and hyperactivity/impulsivity, there were no other genetic influences shared by reading and either ADHD symptom dimension. These results suggest that comorbidity between reading difficulties and ADHD is primarily attributable to common genetic influences that lead to slow PS.

RD and MD

RD and MD co-occur in 30–70% of individuals with either disorder, again a higher-than-chance overlap (Badian, 1999; Kovas et al., 2007; Landerl & Moll, 2010). Twin studies suggest that some of the same genetic influences contribute to both disorders (Light & DeFries, 1995; Plomin & Kovas, 2005). However, relatively few studies have examined possible shared cognitive predictors.

The existing literature suggests that MD is associated with a pronounced weakness in numerosity, the understanding of different conceptual properties of numbers (Cirino, Fletcher, Ewing-Cobbs, Barnes, & Fuchs, 2007; Geary, Hoard, Byrd-Craven, Nugent, & Numtee, 2007). In addition, some previous studies have reported that individuals with MD have weaknesses in PA (Geary et al., 2007), WM (Passolunghi & Cornoldi, 2008; Raghobar, Barnes, & Hecht, 2010), set shifting (van der Sluis, de Jong, & van der Leij, 2004), and PS and NS (Andersson & Lyxell, 2007; Geary et al., 2007). Since several of these cognitive weaknesses have also been implicated in RD, a logical step is to investigate which might help account for the overlap of the two disorders.

A few studies have now investigated unique and shared cognitive predictors of RD and MD. Using multiple regression models, Willcutt and colleagues (2013) reported that PA and NS were unique predictors of RD, while set shifting uniquely predicted MD. The two disorders shared weaknesses in WM, PS and verbal comprehension (VC).

Cirino and colleagues (2013) also tested the association of RD and MD with various cognitive predictors, including language skills (PA, NS and vocabulary), WM, PS, and foundational numerical competencies. PA predicted RD while PS, non-verbal problem solving and foundational math competencies predicted MD. WM predicted both disorders. PS was assessed using a speeded perceptual discrimination task that had reduced linguistic and graphomotor demands, which may explain the lack of association with RD.

Moll and colleagues (2014) investigated a slightly different set of cognitive risk factors that included PS, WM, and temporal processing (a time reproduction task). The authors argued that the predictors were chosen because all three have been associated with ADHD, which is also comorbid with both RD and MD. Results of this study converged with Cirino et al. (2013), with VWM being associated with both disorders. PS and NS predicted RD uniquely, while temporal processing and visuospatial WM predicted MD.

Across these studies, there is convergence that WM may be an important shared cognitive deficit contributing to the comorbidity between RD and MD. Results are more variable for PS, partly depending on the specific tasks used. These results highlight that specific model results can vary depending on the constructs included, the tasks used to assess each construct, and sample characteristics (i.e., a population sample versus a learning disabilities sample).

MD and ADHD

Fewer studies have investigated the comorbidity between MD and ADHD. In a study of 476 children with ADHD, Capano et al. (2008) found a prevalence rate of 18.1% for comorbid

MD + ADHD. There was no effect of age, sex, ADHD subtypes or comorbid conduct disorder on the frequency of MD. Children who had both disorders attained lower IQ, language, and academic scores compared to children with ADHD alone. Children who had ADHD, MD and RD were the most impaired, and had distinct deficits in receptive and expressive language skills.

Twin studies have shown that genetic factors account for a significant portion of the comorbidity between MD and ADHD (Hart et al., 2010; Polderman et al., 2011). Additionally, as has been shown with RD, there is a stronger phenotypic association between MD and inattention than between MD and hyperactivity/impulsivity (Rodriguez et al., 2007). Since both ADHD symptoms and math ability have also been shown to be genetically associated with reading and general cognitive ability (Hart, Petrill, Thompson, & Plomin, 2009), the association between ADHD and math could possibly be confounded by the genetic factors shared with reading or IQ. However, Greven et al. (2014) found that the genetic association between inattention and math ability could only be partially accounted for by reading and IQ. Thus, there are likely to be unique as well as shared influences among the three disorders.

To date, no study has specifically investigated potential cognitive risk factors that could account for the comorbidity between MD and ADHD. Additionally, there has been no study that has attempted to model all three symptom dimensions (reading, math, and attention) at once to test which cognitive predictors may be unique and which may be shared by two or more disorders. The current study aims to fill these gaps in the literature. Because of the evidence described above that the inattention symptom dimension of ADHD is more strongly related to reading and math than is hyperactivity/impulsivity, we focused only on the former symptom dimension in the current investigation. It is also important to place these models in a developmental context. Although there is evidence to suggest that core deficits likely persist across development in conditions such as RD, MD, and ADHD, it is not clear whether the relative importance of risk factors remains constant over time. In fact, the reading literature would suggest that this is likely not the case. Constructs such as PA, semantic knowledge, and NS may predict different amounts of variance in reading depending on a child's stage of reading acquisition (i.e., early literacy where accurate decoding is emphasized vs. later stages of reading acquisition where reading fluency and comprehension is emphasized) (de Jong & van der Leij, 2002). This issue extends to the modeling of comorbidity, and as such, it will be important to test these models at different ages.

Hypotheses—We addressed several specific questions in the current study. First, we asked whether a phenocopy phenomenon could possibly explain the symptom overlap among reading, math, and attention. It is possible that poor attention more globally impacts children's ability to acquire academic skills, and conversely, that good attention supports acquisition of both reading and math. We thought this possibility was unlikely to account for most of the overlap among reading, math, and attention based on previous literature showing that the comorbidity between reading-related and attention problems emerges early in development (Boetsch, 1996) and arises from shared cognitive and etiologic risk factors (Ebejer et al., 2010), but thought it important to explicitly test in this data set. Thus, we

hypothesized that attention would not fully account for the overlap between reading and math.

Second, we tested a multiple cognitive predictor model of the overlap among individual differences in reading, math, and attention. We developed specific hypotheses based primarily on previous results from this sample, with additional consideration for the broader learning disorders literature reviewed above. First, we predicted that PS would be a significant predictor of all three outcomes, and would help explain all three pairs of symptom overlap. Second, we expected that VC would additionally help account for the overlap between reading and math. Third, we expected to find cognitive predictors unique to each outcome. Specifically, we hypothesized that phonological awareness (PA) and NS would uniquely predict reading and inhibition would uniquely predict attention. For math, we predicted that measures of nonverbal problem solving (in our sample, captured by the Wechsler perceptual organization (PO) factor) and WM would be unique predictors. Specific measures of numerosity were not available in this sample, unfortunately. In a follow-up analysis, we tested an alternative model to see if executive functions (WM and inhibition) could account for the comorbidity among symptom dimensions. Thus, we allowed these cognitive factors to predict all three outcomes and evaluated whether inclusion of these additional paths improved model fit relative to our first hypothesized model. There is empirical support for relationships between reading-WM, attention-WM, and math-inhibition in the broader literature. However, previous studies in the current sample have not found these associations to be significant after accounting for the other cognitive factors included in this study.

Method

Participants

Participants included a total of 636 children and adolescents (305 males and 331 females), 8–16 years. The participants were recruited as part of the Colorado Learning Disabilities Research Center (CLDRC) twin study, which is an ongoing population-based study of the etiology of learning disorders, described elsewhere (DeFries et al., 1997; Willcutt et al., 2005). In brief, permission was sought from parents of all twin pairs between 8–18 years in 22 local school districts to review school records. If either member of a twin pair had a history of reading or attention difficulties, the pair and any siblings were invited to participate in the study. A comparison group of control twins was selected from the overall sample of pairs who did not meet the screening criteria for learning problems. Inclusion criteria included the following: (1) English-speaking home, (2) no evidence of neurological problems or history of brain injury, (3) no uncorrected visual or auditory deficits, and (4) no known genetic disorders or syndromes. Additional criteria specific to this study were (1) a Full Scale IQ of at least 70 on the Wechsler Intelligence Scale for Children-Revised (WISC-R; Wechsler, 1974) and (2) age range between 8–16 years to minimize missing data due to test version differences associated with age. To preserve the statistical assumption of independence, one twin was randomly chosen from the twin pair, regardless of diagnostic status, to be included in the analyses for this study. Further details regarding the sample demographics are provided in Table 1.

Procedures

The study protocol was approved by the Institutional Review Boards at the University of Colorado, Boulder and the University of Denver. After obtaining informed consent at both institutions, two testing sessions were completed at the University of Colorado, and a third testing session was scheduled approximately one month later at the University of Denver. Participants taking psychostimulant medication were asked to withhold medication for 24 hours prior to each testing session.

Measures

Detailed descriptions of the tasks administered have been published in previous reports (Gayan & Olson, 2001; Willcutt et al., 2005). As a result, Table 2 is limited to a brief description of the tasks grouped according to construct along with a basic definition of each cognitive construct. Descriptive statistics for the overall sample for all measures with standardized scores available are provided in Table 3. As can be seen, the sample performed within the average range overall although slightly above the population mean on most measures. These higher scores may reflect a Flynn effect in part, since several older tests are used in the CLDRC to maximize consistency in measures across many years. Similar to other volunteer research studies, there may also be some ascertainment bias contributing to this effect. All models utilized continuously distributed scores of the cognitive and symptom dimensions. A dimensional Attention score was calculated for parent and teacher raters by averaging the ratings on the nine DSM-IV inattention items on the Disruptive Behavior Rating Scale (DBRS; Erford, 1993).

Data Cleaning

Raw scores from the tasks in Table 2 were used in the analyses and were reflected, when necessary, so that higher scores were associated with better performance. Outliers were winsorized to 4 SD, and variables were checked for extreme departures from normality (Kline, 2005). We controlled for possible linear and nonlinear effects of age by regressing the raw scores on age and age squared and saving the unstandardized residuals for further analyses.

For the cognitive indicators, missing data was minimal. For the symptom dimensions, missing data was minimal for the indicators of the Single Word Reading and Math factors (<1% missing). For the Attention symptom dimension, it was more common to have missing data because information was gathered from multiple raters: mothers (4% missing), fathers (22% missing), and teachers (16% missing).

Analyses—SEM analyses were run with AMOS 22.0 using maximum likelihood estimation and imputation of missing data using full information maximum likelihood estimation. We used the following general guidelines for reasonable model fit: $\chi^2/df < 3$, Comparative Fit Index (CFI) > .90, Root Mean Square Error of Approximation (RMSEA) < .08 (Kline, 2005).

As described in the hypotheses section above, we planned to compare two multiple predictor models: a first model in which specific hypothesized cognitive predictor-symptom

relationships were based heavily on previous results in the current sample, and a second model emphasizing the contribution of executive functioning skills to symptom overlap. After identifying a model that fit the data well, we examined how well the model accounted for comorbidity by examining the correlations among the reading, math, and attention error terms. The logic was that if the included cognitive predictors fully explain symptom overlap, the correlations among any residual symptom variance (i.e., the error terms) should be null. Finally, we tested more specifically which shared cognitive predictors could account for comorbidity by dropping them, one at a time, from the final model. The logic in this case was that if a given predictor accounted for symptom covariance/comorbidity, when that predictor is removed from the model then the correlation between symptom error terms should increase again.

Results

Measurement Models

Symptom dimensions—Our measurement model for the symptom dimensions of reading, math, and attention is displayed in Figure 1. The latent factors represent continuously distributed symptom dimensions underlying the diagnostic categories of the related disorders (RD, MD, and Inattentive ADHD). Our proposed model fit the data well ($\chi^2(17)=50.48$, $p < .001$; $\chi^2/df=2.97$; CFI=.99; RMSEA=0.06) and was accepted without further modification. The significant correlations among all three symptom dimensions represent the continuous comorbidities that we will seek to explain with the cognitive dimensions using structural equation models.

Cognitive dimensions—Our proposed model for the cognitive dimensions of VC, PO, PA, VWM, NS, PS, and inhibition (Inhib) is displayed in Figure 2 and Table 4. Again, the model fit the data well ($\chi^2(253)=663.41$, $p < .001$; $\chi^2/df=2.62$; CFI=.94; RMSEA=0.05) and was accepted without further modification.

Phenocopy Hypothesis

Next, we evaluated whether the symptom overlap might arise from a phenocopy phenomenon in which attention impacts the acquisition of academic skills across domains. We tested a very simple model in which the latent trait of attention predicted the latent traits of both reading and math. Error terms for the reading and math traits were correlated. The key question was whether this model would significantly reduce the correlation between these error terms compared to the zero-order correlation of .77 (as shown in Figure 1). Fit statistics for this model were good ($\chi^2(17)=15.48$, $p < .001$; $\chi^2/df=2.97$; CFI=.99; RMSEA=0.06). However, the correlation between reading and math error terms was essentially unchanged ($r = .73$, $p < .001$). This result is consistent with our hypothesis and suggests that the three-way symptom overlap does not primarily arise from a phenocopy phenomenon. Instead, it appears that cognitive constructs are needed to explain the overlap among reading, math, and attention.

Multiple Deficit Models

Full Group—Next, we tested a comorbidity model in the full sample in which cognitive dimensions predicted symptom dimensions. We began with a hypothesis-driven model based on the overall learning disorders literature as well as on previous results in this sample. Specifically, we allowed VC, PA, PS, and NS to predict reading; VC, PO, VWM, and PS to predict math; and VC, PO, PS, and Inhibition to predict attention. Model fit was good ($\chi^2(458)=1153.53$, $p < .001$; $\chi^2/df=2.52$; CFI=.94; RMSEA=0.05). Results were largely consistent with our predictions and with prior results, with a few exceptions. PS did not explain significant unique variance in reading. Furthermore, PO did not explain significant unique variance in any outcome. We thought these findings might at least partly reflect the construct overlap between PO and PS, given that the PO subtests are completed under time pressure. We therefore tested a second model that did not include PO and was otherwise identical to the first. Model fit remained good ($\chi^2(348)=907.94$, $p < .001$; $\chi^2/df=2.61$; CFI=.95; RMSEA=0.05) and consistent with considerable previous literature, PS now explained significant unique variance in reading. We therefore accepted this second model, which is shown in Figure 3.

Several aspects of this model are worth highlighting. Each symptom is predicted by multiple cognitive dimensions, some of which are shared across symptoms (PS predicting reading, math, and attention; VC predicting reading and math) and some of which are unique to a particular symptom (PA and NS for reading; VWM for math; and inhibition for attention). The cognitive predictors fully explained the comorbidity among the symptoms; correlations among all sets of error terms became nonsignificant. The cognitive predictors explained 79%, 88%, and 28% of the variance in reading, math, and attention, respectively.

To test which specific predictors explained the comorbidity among symptoms, we dropped PS from the final model, then dropped VC from the final model. Without PS as a predictor, the correlation between the reading and attention error terms became significant ($r=.13$, $p=.03$), as did the correlation between the math and attention error terms ($r=.18$, $p=.04$). However, the correlation between the reading and math error terms remained close to 0 and nonsignificant. In contrast, when VC was dropped from the full model, the correlation between the reading-attention and math-attention error terms remained nonsignificant, but the correlation between reading and math became large and statistically significant ($r=.46$, $p < .001$). Overall, these results suggest that PS accounts for much of the overlap of attention with both reading and math, but that the overlap of reading and math owes to VC.

Next, we evaluated an alternative model to test whether executive functions (working memory and inhibition) helped account for the comorbidity among symptoms. To the model in Figure 3, we added paths allowing VWM to predict both reading and attention, and paths allowing inhibition to predict both reading and math. Addition of these paths did not significantly improve model fit ($\chi^2(4)=2.97$, $p > .4$), and none of the new paths was statistically significant. This model provided no evidence that these executive functions helped account for the overlap among symptoms.

We conducted a follow-up analysis to better understand the relationship of verbal skills to math achievement. We wondered whether this relationship was carried mainly by a link

between VC and PIAT Math, which includes many word problems. In contrast, our other math measure (WRAT-R Math) emphasizes computation. We therefore ran the final model shown in figure 3, but using only WRAT-R Math (treated as an observed variable) as the math outcome. Model fit remained good ($\chi^2(322)=844.94$, $p < .001$; $\chi^2/df=2.62$; CFI=.95; RMSEA=0.05), and the overall pattern of results was very similar to that reported above. Specifically, VC, PS, and VWM all continued to be significant predictors of math outcome (with standardized path weights of .33, .20, and .33), and the overlap among all three symptom dimensions remained nonsignificant. The cognitive predictors explained 54% of the variance in WRAT-R math. It is expected that the proportion of variance explained will decrease somewhat, since the observed variable includes error variance.

Multiple Groups Analyses

We formally tested whether the final model varied as a function of age using multi-group SEM analyses. We compared groups based on a median age split. There were 325 younger children (<10.33 years) and 315 older children (≥ 10.33 years).

First, we tested whether measurement weights could be constrained to be equal across the two groups without significant loss of fit. Results indicated significant model differences across age groups ($\chi^2(20)=88.42$, $p < .001$). To understand this finding, we evaluated each measurement weight individually for age group differences. We applied a Bonferroni correction for multiple testing. Seven of 29 individual weights were significantly different in younger versus older children, including observed variables that loaded on VC (Information, Similarities), PA (Phoneme Deletion 1 and 2, Pig Latin), and Math (PIAT Math and WRAT-R Math). Although the differences in unstandardized weights for these variables were statistically different in the two groups, the magnitude of the differences was generally small. Furthermore, the standardized loadings were very similar in every case, because the direction of variance differences aligned with the direction of the loading differences.

Next, we tested whether structural weights could be constrained to be equal across the two groups without significant loss of fit. Results again indicated significant model differences across age groups in the omnibus test ($\chi^2(10)=24.95$, $p=.005$). However, none of the individual structural weights significantly varied in older vs. younger children after correction for multiple comparisons.

Finally, we constrained the correlations among the error terms for the symptom dimensions (representing any remaining covariance after accounting for shared cognitive predictors) to be equal across older and younger groups. There was no evidence that these relationships varied as a function of age ($\chi^2(3)=1.00$, $p=.80$).

Discussion

The goal of the current study was to better understand the basis for the overlap among word reading, mathematics skill, and attention across the full range of individual differences in a community-based sample of twins aged 8 to 16. These three skills are moderately to highly correlated, and the corresponding neurodevelopmental disorders of RD, MD, and inattentive ADHD co-occur at greater-than-chance levels. We asked which cognitive skills underlie

these relationships. As predicted, results did not support a phenocopy hypothesis in which low reading and math achievement both arise from poor attention. Instead, results were consistent with a multiple deficit or multiple predictor framework in which the shared variance among each pair of symptoms could be attributed to a single common cognitive predictor. Also consistent with the predictions of a multiple deficit/multiple predictor framework, each symptom also had at least one unique predictor that was not shared with other outcomes. Important novel findings included that verbal-conceptual skills specifically contributed to the overlap between reading and math, while PS contributed to the overlap between math and attention.

As expected based on previous work in this sample, PS helped account for the overlap between reading and attention. Since PS weaknesses have also been linked to MD, we had initially hypothesized that this single cognitive factor might contribute to overlap among all three symptom dimensions. This hypothesis was partially supported. PS did explain significant variance in each symptom outcome, and accounted for the overlap between math and attention. However, VC rather than PS explained the strong relationship between reading and math achievement. This pattern continued to be evident when math was assessed with a single measure that emphasized calculation skills rather than word problems. Unique predictors for each symptom were consistent with previous research and included PA and NS for reading, VWM for math, and response inhibition for attention.

Together, cognitive factors accounted for the large majority (approximately 80–90%) of the variance in reading and math skills. As expected, variance accounted for in attention was much lower (<30%). Even this figure is at the higher end of variance in attention that researchers have been able to explain with neuropsychological tasks, likely because we modeled attention as a latent trait that removed error variance. The consistent failure across studies to account for more variance in attention probably arises from several factors, including: 1) method variance (of the three outcomes and six predictors included in Figure 3, eight are assessed with objective cognitive tasks while only attention is assessed with rating scales); 2) restricted variance at the high end of the scale (like most attention rating scales, those used in the current study are designed to measure problematic behaviors but have limited ability to detect individual differences at the other end of the spectrum); and 3) perhaps a less mature neuropsychological theory of inattentive ADHD in comparison to MD and certainly to RD.

As illustrated by the current special issue, there is growing interest in the role that executive functions play in academic skill development. One hypothesis is that executive functions contribute in a general fashion to achievement across skill domains, and thus help account for comorbidity or covariation among symptom dimensions. The current study provided no evidence to support such a hypothesis. Instead, we found that specific executive functions differentially related to certain outcomes (i.e., VWM to math and inhibition to attention), but did not help account for the overlap between any two symptoms. Two important caveats are: 1) The current study emphasized basic reading skills, while our math composite tapped a mix of basic and complex skills. A very different pattern could emerge with more detailed measurement of higher-level skills such as reading comprehension, math problem solving, and written composition; and 2) Model results can vary substantially depending on which

predictors are included, and as made clear in the introduction, exactly which tasks are used to assess those constructs. Thus, for example, if we had eliminated PA and NS or if we had used speeded measures emphasizing only perceptual speed or more basic reaction time rather than graphomotor speed, it is possible that VWM could have appeared to serve as a shared predictor of reading and math. A related point is that several of the cognitive constructs included in the current study were derived in part or in full from one of the Wechsler instruments, and some of them load strongly on Spearman's "*g*" or a general intelligence factor. Of course, essentially all cognitive or neuropsychological tests correlate with *g* at least to an extent (Pennington, 2008), so this interpretive issue is not unique to measures that are given as part of an IQ test. We obtained different patterns of results for VC, PO, PS, and VWM, which suggests that there is at least some specificity to the relationships between these cognitive factors and symptom domains. However, we did not explicitly test the extent to which common cognitive variance or *g* contributes to symptom covariance and this is an important question for future research that will require a different methodological approach.

The large age range included in the current study allowed us to test whether the relationships between cognitive predictors and symptom dimensions shifted as children grew older. The overall pattern of relationships was quite similar in younger and older participants. We found a few small but statistically significant differences in measurement aspects of the model. However, there was not compelling evidence that cognitive variables differentially related to symptom outcomes or to comorbidity in younger versus older children. Of note, however, our older and younger subgroups still included significant age variance. It is possible that results could look different in a very large sample allowing for comparison of results across narrower age bands, or in individuals younger or older than those included in the current study.

Results have several implications. Because presence of comorbid learning disorders is associated with increased functional impairment (Willcutt et al., 2013), early identification of children at risk for more than one disorder is especially important. The current study adds to a growing body of evidence suggesting that weak processing speed is a risk factor for multiple poor outcomes. Thus, the educational and clinical utility of very early screening for processing speed deficits should be investigated by future research. Similarly, early screening for children with poor verbal skills could likely identify those at risk for academic difficulties across the curriculum. The current finding of a similar pattern of results across age groups, in conjunction with previous work showing very high stability of individual differences in academic skills over time (Hulslander, Olson, Willcutt, & Wadsworth, 2010) suggests that school age assessment should predict adolescent cognitive and educational profiles fairly well. However, group findings never generalize perfectly to every individual in the distribution, so individual classification and prediction is inevitably challenging (Pennington et al., 2012). Thus, for educational and clinical decision making at the individual level (e.g., determining whether a particular child qualifies for continued services), periodic re-evaluation is likely to remain appropriate.

Limitations

These results should be interpreted in the context of several limitations. First, our cross sectional sample and study design do not allow us to draw firm conclusions about causality. Working within the paradigm of developmental neuropsychology, we assume that discrete cognitive processes underlie, or cause, observed symptoms. However, it is also possible that the causal direction runs the other way, or that there are bidirectional relations. In fact, empirical evidence supports such a bidirectional relationship between reading and PA (Morais, Cary, Alegria, & Bertelson, 1979) as well as reading and vocabulary (one part of VC) (Stanovich, 1986).

Second, as previously mentioned, interpretation of results can be heavily influenced by the variables that are included in the model. Readers of some of our previous studies have been surprised that VWM does not play a more important role in the prediction of reading, for example. However, in the current sample, we have consistently found that when both PA and VWM are included, PA “wins” the competition and VWM does not explain significant *unique* variance in reading. Of course this does not mean that reading does not involve VWM. As is evident in Table 4, all the cognitive predictors overlap to some extent with one another, and PA tasks certainly also tap into working memory skill to a degree.

A third important limitation is that we tested our model across the full range of individual differences and not only in a disordered sample to maximize variance and sample size. Based on evidence that essentially all behaviorally-defined developmental disorders represent the low tail of a continuously distributed symptom dimension (Pennington, 2014), we think results have important implications for understanding the comorbidities among the common and impairing disorders of RD, MD, and ADHD as well. However, a somewhat different pattern of results could emerge in a sample more heavily selected for learning disabilities.

Future directions

In sum, results provide support for a multiple cognitive predictor model of three domains important in children’s educational achievement: reading, math, and attention. Findings suggest several important directions for future research. One set of questions concerns the mechanisms through which shared cognitive predictors influence multiple outcomes. For example, some researchers have proposed that slowed PS serves as a “bottleneck” that limits children’s abilities to master various skills (Dennis, 2000). Alternatively, PS may simply serve as a proxy for some other variable, such as white matter integrity (Penke et al., 2010), that influences performance across domains. Similarly, why do verbal skills underlie the relationship between reading and math achievement? Is it because so much teaching takes place in a verbal context or some other reason more internal to the child? As noted in the limitations sections above, it will also be important to test related models in longitudinal samples that include measures of both basic and complex academic skills. Future studies should also elucidate links to other levels of analysis, including brain bases and etiologic risk and protective factors. Finally, the potential relevance of these findings for early identification and remediation of children at risk for multiple disorders will need to be rigorously evaluated by future studies.

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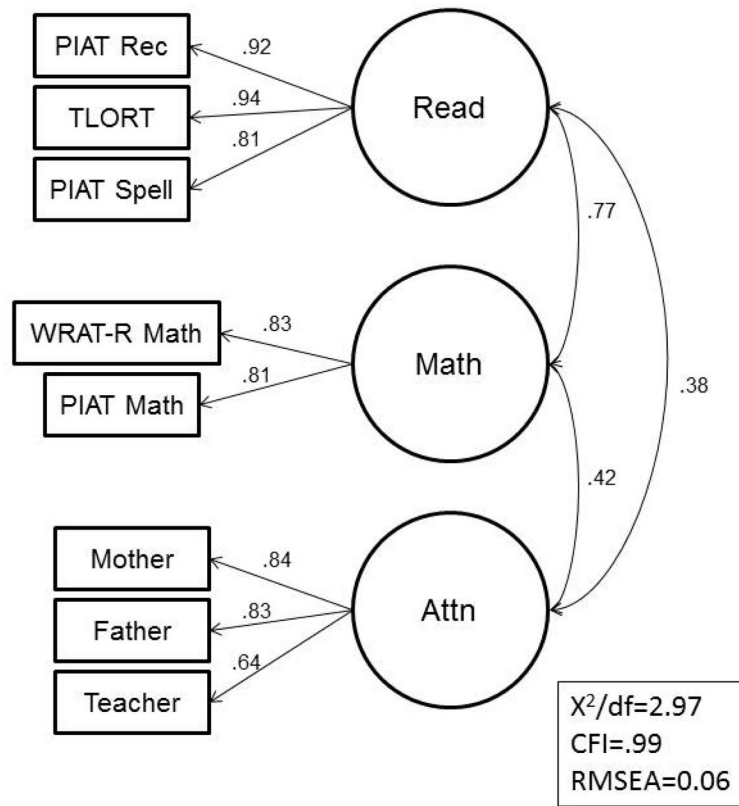


Figure 1.
 Measurement model of symptom dimensions.
 Notes. Read=Single word reading; Attn=attention; PIAT Rec= PIAT Reading Recognition;
 TLORT=Time Limited Oral Reading Test.
 Standardized weights shown.

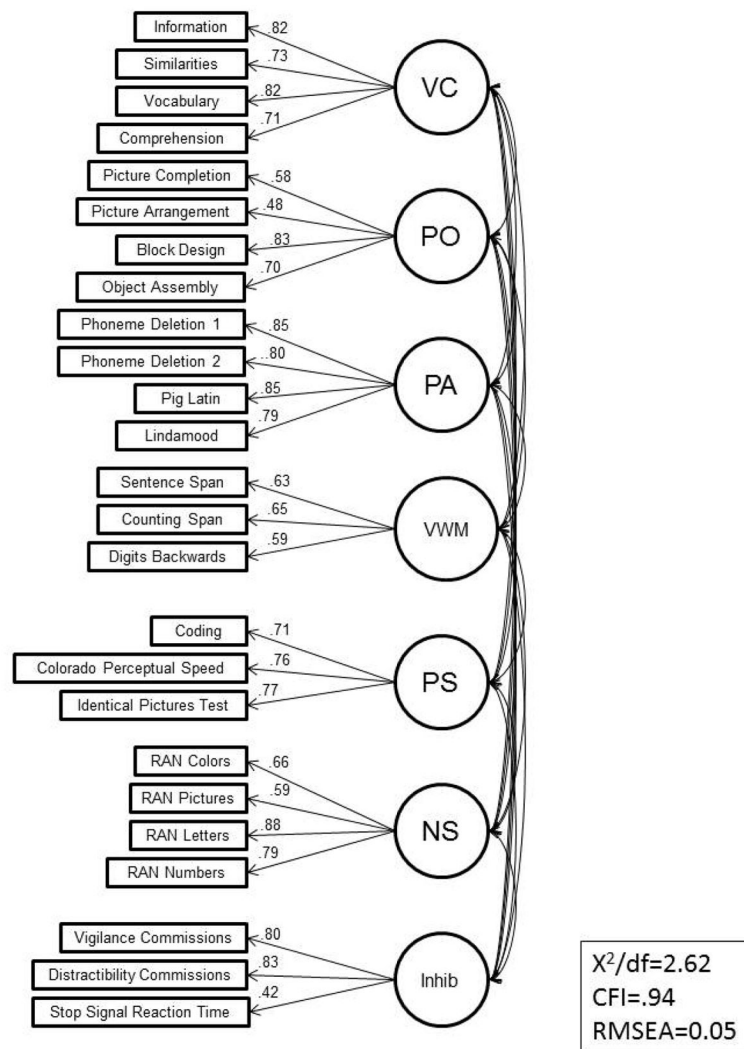


Figure 2.
Measurement model of cognitive dimensions.

Notes. VC=Verbal Comprehension; PO=Perceptual Organization; PA = Phonological Awareness; VWM=Verbal Working Memory; PS=Processing Speed; NS=Naming Speed; Inhib=Inhibition.

Standardized weights shown.

For ease of reference, correlations among latent constructs are shown in Table 4.

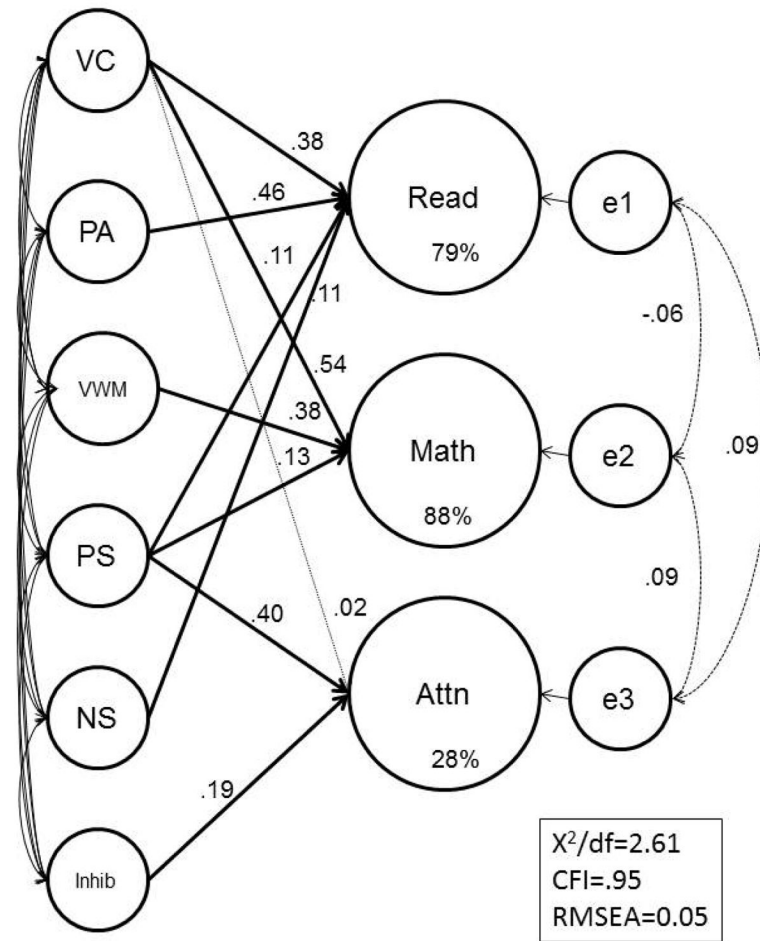


Figure 3.
Comorbidity model in full sample.
Note. Standardized weights shown. Solid paths are statistically significant at the $p < .05$ level. Dotted paths are not statistically significant ($p > .05$).

Table 1

Sample characteristics

Sample size	n = 636
Males: n (%)	305 (48.0)
Age in years: Mean (SD)	10.94 (2.39)
Mother years of education: Mean (SD)	15.04 (2.44)
Race: % Caucasian	82.0
WISC-R Full Scale IQ: Mean (SD)	108.02 (13.57)

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Table 2

Indicators for the symptom and cognitive dimensions. Basic definitions of the cognitive dimensions are provided for reference.

Measure	Reliability	Reference	Brief Description
Symptom dimensions			
<i>Single word reading</i>			
PIAT Reading Recognition	.89	(Dunn & Markwardt, 1970)	Read single words that increase in semantic and phonetic difficulty.
PIAT Spelling ^a	.65	(Dunn & Markwardt, 1970)	Select the correct spelling of a spoken word from 4 phonologically similar options.
Time-Limited Oral Reading	.89	(Olson, Wise, Connors, Rack, & Fulker, 1989)	Read aloud single words within 2 seconds of their presentation.
<i>Mathematics</i>			
PIAT Math	.90–.96	(Dunn & Markwardt, 1970)	Solve multiple-choice math problems that are read aloud by examiner (main concepts, with some computations)
WRAT-R Math	.92	(Jastak & Wilkinson, 1984)	Solve paper-and-pencil computations
<i>ADHD inattentive symptoms</i>			
Disruptive Behavior Rating Scale	.71 – .94 ^b	(Erford, 1993)	Mother, father, and teacher ratings of DSM-IV inattention symptoms on a 0 to 3 scale with anchors not at all, often, sometimes, and very often
Cognitive dimensions			
<i>Verbal comprehension (VC)</i> Language-based knowledge and reasoning.			
WISC-R Vocabulary	.86	(Wechsler, 1974)	Define words aloud.
WISC-R Similarities	.81	(Wechsler, 1974)	Explain how two objects or concepts are alike.
WISC-R Information	.85	(Wechsler, 1974)	Answer common knowledge questions.
WISC-R Comprehension	.77	(Wechsler, 1974)	Describe solutions to real-world questions or problems.
<i>Perceptual organization (PO)</i> Novel, nonverbal and spatial reasoning.			
WISC-R Block Design	.85	(Wechsler, 1974)	Recreate pictured designs with colored blocks.
WISC-R Picture Completion	.77	(Wechsler, 1974)	Identify the missing part of a picture.
WISC-R Picture Arrangement	.73	(Wechsler, 1974)	Put pictures in order to tell a story.
WISC-R Object Assembly	.70	(Wechsler, 1974)	Complete object puzzles.
<i>Phonological awareness (PA)</i> Oral language skill characterized by the ability to dissect a spoken word into smaller sound units, the smallest of which are phonemes.			
Phoneme deletion	.80	(Olson, Forsberg, Wise, & Rack, 1994)	Remove a phoneme from a word or nonword and say the resulting word
Lindamood Auditory Conceptualization Test	.67	(Lindamood & Lindamood, 1979)	Use colored blocks to represent phonemes in sound sequences and nonwords
Pig Latin	.78	(Olson et al., 1989)	Move the first phoneme to the end of the word, then add “ay.”
<i>Verbal working memory (VWM)</i> The ability to hold verbal information in mind while simultaneously performing a manipulation or a separate cognitive task.			
WISC-R Digit Span Backward	.78	(Wechsler, 1974)	Repeat strings of numbers of increasing length in reverse order.
Sentence span	.65–.71	(Kuntsi, Stevenson, Oosterlaan, & Sonuga-Barke, 2001)	Provide the last word for a set of simple sentences read by the examiner, then reproduce these words in order after the set is completed.

Measure	Reliability	Reference	Brief Description
Counting span	.55–.67	(Kuntsi et al., 2001)	Count aloud the number of yellow dots on a series of cards. At the end of each set state in order the number of yellow dots that appeared on each card in the set.
<i>Naming speed (NS)</i>			The ability to rapidly recognize and name a restricted set of well-known visual items presented in a series.
RAN Colors	.82	(Denckla & Rudel, 1976)	Name colors as quickly as possible for 15 seconds.
RAN Numbers	.86	(Denckla & Rudel, 1976)	Name numbers as quickly as possible for 15 seconds.
RAN Letters	.86	(Denckla & Rudel, 1976)	Name letters as quickly as possible for 15 seconds.
RAN Pictures	.80	(Denckla & Rudel, 1976)	Name picture as quickly as possible for 15 seconds.
<i>Processing speed (PS)</i>			Mental efficiency of processing and matching symbols, such as letters, numbers, and pictures.
WISC-R Coding	.72	(Wechsler, 1974)	Rapidly copy novel symbols associated with numbers based on a key
Colorado Perceptual Speed Test Parts 1 & 2	.81	(Decker, 1989)	Identify a target string of letters or letters and numbers among three foils. Letter strings are not pronounceable.
Identical Pictures Test	.82	(French, Ekstrom, & Price, 1963)	Identify a target picture among an array of pictures with four foils.
<i>Inhibition (Inhib)</i>			Ability to stop a prepotent motor response under specific conditions.
Gordon commission errors	.72 – .85	(Gordon, 1983)	Total responses to incorrect targets during a continuous performance test in two conditions (with and without distractors).
Stop Signal Reaction Time	.90 – .96	(Logan, Schachar, & Tannock, 1997)	Computerized variation of a go/no-go task.

^aPIAT Spelling is included under the single word reading construct because is essentially a single word reading task involving spelling recognition, not production.

^bRange includes estimates of 1-year test-retest reliability and Cronbach's alpha

Table 3

Descriptive statistics for cognitive and symptom variables with standardized scores available.

Variable	Score Type	Mean (SD)
PIAT Reading Recognition	SS ^a	103.63 (12.61)
PIAT Spelling	SS	101.84 (12.79)
PIAT Math	SS	106.39 (14.38)
WRAT-R Math	SS	97.36 (17.13)
WISC-R Full Scale IQ	SS	108.02 (13.57)
WISC-R Information	ss ^b	11.09 (2.93)
WISC-R Similarities	ss	12.53 (3.03)
WISC-R Vocabulary	ss	11.17 (2.84)
WISC-R Comprehension	ss	11.81 (2.96)
WISC-R Picture Completion	ss	11.56 (2.52)
WISC-R Picture Arrangement	ss	11.17 (2.52)
WISC-R Block Design	ss	11.55 (3.11)
WISC-R Object Assembly	ss	10.49 (2.89)
WISC-R Coding	ss	9.65 (3.00)
WISC-R Digit Span	ss	10.19 (2.95)

Notes.

^aStandard Score with mean = 100 and standard deviation = 15.^bScaled score with mean = 10 and standard deviation = 3.

Table 4

Correlations among latent cognitive constructs.

	VC	PO	PA	VWM	PS	NS	Inhib
VC	1						
PO	.61	1					
PA	.64	.47	1				
VWM	.63	.53	.70	1			
PS	.55	.52	.46	.57	1		
NS	.38	.16	.51	.45	.57	1	
Inhib	.35	.28	.50	.47	.43	.33	1

Notes. VC=Verbal Comprehension; PO=Perceptual Organization; PA = Phonological Awareness; VWM=Verbal Working Memory; PS=Processing Speed; NS=Naming Speed; Inhib=Inhibition.

All correlations are significant at p<.001 level, except PO-NS correlation p-value = .002.